CHRONIC TUBERCULOSIS OF THE KIDNEY.[1]

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This subject was selected for my paper of the evening because it seemed to me that it would make an appeal to a general audience. I welcomed the opportunity to present a review of this subject to you, as my experience has shown me that the profession does not fully appreciate the clinical picture of renal tuberculosis and the great importance of its earliest recognition, when still localized to one kidney. Before an audience of this character I naturally shall not go in great detail into many of the refinements in diagnosis and treatment that have developed in recent years, and even if I fail to tell you anything particularly new, I feel sure I shall have reawakened your interest in the subject under discussion and perhaps helped you to recognize cases of this disease.

There has been a growing tendency to call experts to make the diagnoses when the patient's symptoms fail to respond to treatment, and ultimate reliance on the expert has undoubtedly interfered with the general practitioner's independent and early recognition of renal tuberculosis. One can readily understand the practitioners' attitude, based as it is on their unwillingness to admit their inability to treat such slight symptoms as most patients present in the early stages of renal tuberculosis. But in fairness to the patients, why do they persist so long in blind treatment of these cases without arriving at a definite diagnosis? In the typical cases the expert should not be required to make the diagnosis, but his work becomes absolutely necessary in deciding which kidney, or whether both are diseased or not.

There is probably no disease which surgery has done more to clarify in every detail than renal tuberculosis. Its pathology, its diagnosis and its treatment have all been placed on a firm foundation by the surgeons; and as it is unfortunately a very frequent renal disease, in fact the most frequent renal disease that requires removal of a kidney, surgery can be proud of its accomplishment. Many of the profession in all lands have contributed to this study. I need mention only such names as Gross, Steinthal, Morris, Guyon, Tuffier, Israel and Brown to emphasize the international coöperation that has helped to clarify our views on chronic renal tuberculosis.

The view that chronic tuberculosis of the kidney usually began as a unilateral affection secondary to some other often inactive focus only gradually gained acceptance. Pathologists who saw the end-picture of this disease, with tuberculous deposits in both kidneys, in the ureters, in the bladder, perhaps also deposits in the prostate, vesicles, epididymes, not to mention more distant foci in the lungs, meninges, joints, bones, etc., were slow to accept the new teachings, based on surgical experience and a few small series of autopsies. Now it has become our daily experience that these kidney deposits are the only active disease, and if a nephrectomy is performed the patient is cured and rid of his active tuberculosis. This marvelous change in the conception of the pathology of chronic renal tuberculosis in contradistinction to acute and subacute miliary tuberculosis is based entirely on surgical experience of the last forty years, and more especially on the work of the last twenty years. With this change in the conception of the disease the importance of early diagnosis became all the greater, and nowadays, with the aid of the cystoscope and the laboratory, suspicious cases are carefully investigated, so that the surgeon is seeing more and more patients in the early stages of renal tuberculosis than ever before. Still even at this date many patients suffer for months before their condition is realized. In going over a series of well over 100 cases I find that nine to twelve months have elapsed from the beginning of the typical symptom-complex until the real condition was definitely diagnosed.

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[1] A Clinical Lecture delivered before the Wayne County Medical Society, Detroit, Mich., January 17, 1921.

Though chronic renal tuberculosis occurs at all ages it is most common in the third and fourth decades, that is between the twentieth and the fortieth year. It is much less common before the twentieth and after the fiftieth years, though we have seen a number of cases in young children and at least 10 cases in patients over the fiftieth year. This agrees fairly well with other large series that have been published except in the slightly greater frequency after the fiftieth year.

As far as sex is concerned males have slightly outnumbered females. In this regard American data seem to vary from those of our European colleagues. Braasch finds males 63½ per cent to 36½ per cent females afflicted with this disease, whereas Wildbolz[2] quoting the figures of European writers, says males are less frequently affected than females in the ratio 1 to 2. If the autopsy data are taken as criteria, rather than the operative data, it would seem that even in Europe the male is more frequently afflicted than the female. The diagnosis formerly was made more readily in the female, and perhaps that accounts for the contradiction between the published incidence in the two sexes. Of the cases that come to operation the right kidney is more often involved that the left, possibly because of its greater mobility. Unfortunately at least 10 per cent of cases come to us when both kidneys are involved in tuberculous disease and at a stage when no radical procedures are justifiable. In this our experience agrees fairly well with published reports (11 per cent to 14 per cent).

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[2] Chirurgie d. Nieren tuberkulose, 1913.

To grasp the symptomatology of chronic tuberculosis of the kidney it is essential to understand the main pathological features of this disease. Practically all cases are hematogenous in origin, being secondary deposits through the blood stream. Very rarely is the involvement ascending and only (in my experience) secondary to prostatic tuberculosis which has led to involvement of the bladder, dilatation of the ureters, followed by ureteral tuberculosis, tuberculosis of the pelvis and kidney or kidneys, the disease being less advanced in the upper urinary tract than lower down. In a large series of cases occasionally one sees a beautiful demonstration of the hematogenous deposit limited to one pole of a kidney, through an accessory artery to which Israel called attention years ago.

For many years there has been considerable dispute as to the primary renal focus, but gradually as surgeons are recovering specimens of earlier and earlier disease it has become more and more evident that in the majority of cases the first manifestation of the disease is in one of the papillæ of the kidney. Whether it starts near the base of the papilla or near its tip cannot be determined, though it would appear that both are favorite sites for the deposit of the tubercle bacillus. Kuester, Oraison, Rovsing, Wildbolz (Wildbolz, p. 26)[3] as well as we have very rarely seen a tuberculosis of the pelvis as primary deposit (L. Buergers[4] case. L. S.). Twenty years ago this was not accepted by investigators, because at that time more advanced cases, cases with extensive caseation, were being operated upon. As the papillary involvement progresses there is extension both into the cortex and into the calices and main pelvis. The bacilli appear to break into the vascular channels as they produce necrosis and caseation, and are carried toward the cortex producing tubercles, and discrete abscesses which both are often found directly under the capsule or separated therefrom by a thin layer of degenerated parenchyma. These shut off depots in the parenchyma of the kidney which do not communicate with the pelvis, probably rarely give symptoms until they perforate into the pelvis or into the perinephric space, which latter is a rare occurrence.

On the other hand the important extension of the papillary tuberculosis is toward the calices, the pelvis, the ureter and the bladder, and in these cases we get the typical symptoms of the disease. Ulcers and tubercles develop in the calices and the pelvis, as well as in the ureter, especially in its upper and lower ends, and finally the bladder is involved in the tuberculous process. With the inflammatory changes in the pelvis, the ureter and the bladder, thickening of the walls of these organs takes place and not infrequently stricture formation with obstruction to the outlet of the kidney secretion.

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[3] v. ante.

[4] L. Buerger.

Not infrequently one sees reparative processes in these tuberculous kidneys, as evidenced by extensive scar formation in a calyx which has shut off an abscess or a tuberculous cyst either near the base of a papilla or in the cortex of the kidney, after destruction of a papilla. In other cases the caseation has gone so far that little or no parenchyma is left and only a pultaceous or partly calcified mass, more or less of kidney shape, persists. If the stricture formation in the ureter is marked, pyonephroses develop, occasionally though rarely only a hydronephrosis. If secondary infection is superadded we see all the changes associated with acute or chronic pyelonephritis added to the picture of renal tuberculosis; occasionally even calculi complicate the picture.

As the process in the parenchyma progresses the perinephric tissues almost regularly undergo inflammatory changes, with or without miliary abscess formation and absorption of perinephric fat. At times a massive perinephritis develops which envelops the kidney in a shell of inflammatory tissue of great density and 1 cm. or more in thickness.

It is evident from this superficial sketch of the pathology that two processes appear to be going on side by side, destruction and protective repair, similar to what is seen in tuberculous disease in other organs. Unfortunately, however, the destructive processes regularly gain the upper hand, and though kidneys are occasionally seen in which extensive calcification or hydronephroses with complete parenchymal absorption have resulted, or empty shells of kidneys which may have discharged their caseated parenchyma through rigid open ureters. these abortive attempts at Nature's cure are so rare that one cannot expect in any given case even such a measure of success from a waiting policy.

As the tuberculous process in the kidney begins usually in the papillary zone the patients generally present a rather uniform symptom-complex which possibly varies with the natural immunity of the patient, expressed in his or her reparative processes as just outlined. No matter what the termination of the disease, whether it end in hydronephrosis, pyonephrosis, stricture of the ureter, perinephritis with abscess, secondary pyelonephritis (non-tuberculous or not), I am under the impression that a careful search of the history will present almost always a rather uniform picture of shorter or longer duration, which is so clear-cut as to be almost diagnostic. When we compare the value of the patient's history of his troubles in gastric or duodenal ulcer with that of the history in the cases under discussion, I am inclined to the opinion that renal tuberculosis gives a more definitely diagnostic train of symptoms than ulcers in the above-mentioned organs.

The striking symptoms of the typical cases are:

1. Frequency (and urgency) of urination.
2. Pain on urination.

3. (*a*) Pyuria (albuminuria).
4. (*b*) Hematuria. Pain in the lumbar region.

These symptoms usually develop insidiously, gradually increasing in severity. Occasionally, however, their development is very sudden—out of a clear sky, as it were, (described later on.)

1. Almost all patients complain of frequency of urination shortly after the beginning of the disease, and though there are occasionally periods of remission, the old symptom recurs to annoy the patient. As these cases often excrete an increased amount of urine their frequency is due in a measure to the polyuria in greater measure, however, to the local irritation of the disease. This annoying frequency may become so marked and the bladder control so impaired that a condition of pseudo-incontinence results. In fact, several cases that have come to us in the young had been under treatment for long periods as cases of enuresis (both night and day). This frequency may be so marked (every ten to fifteen minutes) as to prevent sleep, and as it is usually associated with marked urgency and pain the patients may suffer absolute torture.

2. The character of the pain on urination as well as its location seems to vary. Many patients complain of a burning or pricking sensation either in the deep urethra or near the meatus; others again have localized the pain at the neck of the bladder. In some the pain may be most marked as the bladder is emptied, when they become excruciating, often bringing tears to the patient's eyes. Anodynes are often required in these cases.

3 (*a*) Pyuria is an early symptom and the freshly voided urine with its finely distributed flocculi of pus cells produces an almost characteristic looking, delicate cloudiness. Whether absolutely clear, brilliant urine, as has been repeatedly declared, can be recovered in cases with involvement of the papillæ or pelvis I doubt, though it can readily be seen that such specimen might readily be voided when the process is not in free communication with the excretory channels. It cannot be emphasized too often that it is only when the tuberculous process extends into the excretory channels that the characteristic symptoms develop. In fact, one often sees extensive renal tuberculosis shut off in the parenchyma which has produced only transient symptoms of bladder irritability, succeeded by apparent perfect health, only to suddenly flare up as a fresh extension into the excretory channels develops with the production of symptoms of violent vesical involvement. In the literature a remarkable stress has been placed on the presence of a premonitory albuminuria. Albuminuria is regularly present when there is a pyuria and its significance is far outweighed by the recognition of the pyuria. Whether a really "premonitory albuminuria exists prior to the pyuria, that is prior to the extension of the disease into the excretory channels, I have not been able to convince myself. Still, it must be admitted that at this stage of the disease such an

albuminuria is easily understandable and all mild persistent and unexplained cases of albuminuria should be regarded with some suspicion.

3 (*b*). Hematuria varies greatly in these cases. There frequently is a slight admixture of blood in the purulent urine and well mixed with the same. Other cases pass a few drops or a small amount of fresh blood at the end of micturition as their bladders contract. Severe renal hematuria, as the first symptom in the disease, is an infrequent occurrence in our cases.

4. Pain in the kidney region, at times radiating along the ureter, has been much less frequently noted in our series than in the earlier published series. Some authors (Wildbolz, etc.) believe that pain in the kidney region antedates the symptoms of cystitis in 43 per cent of cases. In looking over the records of the more intelligent patients it is quite surprising how regularly they failed to refer any of their symptoms to the kidney region at the stage in which the bladder symptoms were their chief complaint.

It is remarkable how regularly case after case of renal tuberculosis complains of these characteristic symptoms and without any extensive questioning describe the above picture of bladder irritation, cloudy urine, perhaps hematuria, perhaps pain in the kidney region. They almost invariably report months of local treatment with no effect, though occasionally slight temporary improvement has been noted, only to relapse into a condition of greater distress than existed before the period of apparent improvement. While these are the typical cases, and while their history should suggest the correct diagnosis, which can be confirmed by the proper urinary examinations, there are numerous atypical cases, most of which undoubtedly commence in the above way and then gradually become quiescent or masked, while others, which is unusual, apparently fail to notice the usual symptoms of bladder irritation or by the time they seek medical aid have forgotten that period of their trouble.

These atypical cases are much more difficult to recognize and often baffle an expert urologist for some time. Some of these cases start as a massive hematuria, with or without ureter colics as the very first symptom, and this may rarely be so severe as to require transfusion and even nephrectomy. On the other hand the severe bleeding may cease and then the regular picture of the typical cases may develop.

In some cases the reparative and thickening processes, especially if they occur in the ureter, exclude the disease from the excretory passages, and after a brief period of vesical irritation all the symptoms cease as either a pyonephrosis or a hydronephrosis develops above the closed-off ureter. If the process becomes closed off by stricture formation at one or more calices a similar cessation of symptoms may ensue, but sooner or later as the disease recrudesces a rupture or an extension into the pelvis takes place and suddenly all the symptoms of the typical cases are in evidence. In still another type where a mixed infection has developed the symptoms of the superadded infection, whether it be Bacillus coli or not, may so dominate the picture that one does not even suspect the diagnosis of tuberculosis until the kidney has been removed.

The same applies to some of the cases in which stone complicates the chronic tuberculosis. Another, a fourth, atypical development is that associated with perinephritis, especially the hyperplastic and suppurative types of perinephritis. Fortunately these are unusual and the diagnosis may not be made until some of the perinephric tissue is placed under the microscope. In all of these atypical cases except those with massive bleeding as the initial symptom one is more liable to feel a kidney tumor than in the typical cases.

Having the history of a typical case, or of a case that has once had symptoms suggestive of renal tuberculosis, the first step in confirming the diagnosis is the search for tubercle bacilli in the catheterized urine. From our experience there is no doubt that in the majority of cases of pyuria due to tuberculosis the tubercle bacilli can be found, if careful and repeated search of the properly stained sediment is made. In very advanced cases with thick purulent urine the tubercle bacilli may be so few and far between that they cannot be found. The literature abounds in papers dealing with the difficulties offered by the presence of the smegma bacillus, but in our experience we have had no trouble in recognizing the typical grouped tubercle bacilli and have not confounded it with the smegma bacillus, which is so rarely if ever seen in the catheterized bladder specimen. In our experience over 70 per cent of the cases showed the bacillus of Koch, and if repeated search had been made the percentage would have been much higher. With the development of cystoscopic interpretation one often makes a definite diagnosis before the search is completed, and in these cases, the kidney having been removed for tuberculosis, further study of the urinary sediment is of no great value and consequently abandoned. In those cases in which the kidney has been excluded (autonephrectomy), or in which urine is brilliant and clear, the inoculation of a guinea-pig may at times recognize the tuberculous character of the process. It must, however, be remembered that a positive guinea-pig reaction is at times missed in cases of renal tuberculosis, and at other times this test is positive when the patients have a tuberculous lesion in other organs. A careful analysis of the significance of tubercle bacilluria is always necessary.[5] On the whole a positive smear is, in our experience, of greater value than a positive guinea-pig test.

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[5] Beer, E.: Significance of Ureteral Tubercle Bacilluria. Am. Jour. Med. Sc., August, 1917, vol. cliv.

Another very helpful point in the diagnosis is the culture of the catheterized bladder specimen in cases of pyuria. As the output of tubercle bacilli may vary from specimen to specimen it is always advisable to culture the urine. A sterile pyuria with acid urine is common in renal tuberculosis, whereas in all other conditions, except perhaps in nephrolithiasis, it is rarely seen.

Whereas the above data when positive practically invariably mean urinary kidney tuberculosis in the female, it must not be forgotten that in the male prostatic tuberculosis may rarely produce a picture closely simulating that of typical cases of renal tuberculosis and tubercle bacilli may be present in the urine. Even an expert may find difficulty in interpreting the findings in such a case.

It will surely be conceded that patients whose symptoms suggest the presence of a renal tuberculosis should have their cases studied in this way, and so far by the general practitioner. Having made the diagnosis of tuberculosis in the urinary tract the need of a urologist of experience becomes evident. Some of these cases, in fact too many, are referred without the above studies to the general surgeon who has had no experience in urological diagnosis, a development of the last twenty-five years, and if there is local tenderness and perhaps a palpable mass in the kidney region, such a case is operated upon forthwith, either a nephrectomy or a nephrotomy being done. To operate on any kidney case without attempting to determine the presence of an adequate second kidney prior to the operation is nowadays absolutely unpardonable. All these cases should be studied carefully by the cystoscopist, who must determine, using gas-oxygen anesthesia if necessary where the disease is, and if in one kidney whether the second is healthy and adequate. It is up to the cystoscopist to rule out prostatic tuberculosis as a source of the tubercle bacilluria and it is up to him to diagnose the exact condition of the diseased kidney and to determine that the second organ is not tuberculous.

Fortunately all these things can be done with great accuracy. Experience has proved conclusively that the lesions in the bladder, the edematous ureteral meatus, its more or less rigid walls, the adjacent polypoid (or pseudopapillomatous) edema, its retraction as the ureter becomes thickened, the ulcers and the tubercles about the involved ureter produce a picture which is sufficiently characteristic to make the diagnosis. In 1907 Rovsing[6] made the statement that occasionally the bladder involvement is about the ureter meatus of the healthy kidney. Personally in hundreds of cases examined by me this has never obtained. In view, however, of this publication it is advisable to collect ureteral specimens whenever possible and confirm the presence of pyuria and tubercle bacilli on the side that is chiefly involved in the bladder disease. The use of indigo carmine is invaluable in this work, not only as an aid in locating the ureters in diseased, contracted bladders, but also as an index of the relative function of the two kidneys; usually the diseased kidney excretes a less concentrated stream of indigo than its healthy fellow. In the strictured ureter cases it may be impossible to collect a specimen on that side and by exclusion of the other kidney whose urine is normal plus the local findings one decides that the tuberculosis is on the strictured side. On many of these cases careful observation will detect a weak little bluish stream trickling out of such a strictured ureter.

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[6] Arch. f. Klin. Chirurg., 1907, lxxxii, 10.

Before this audience it is scarcely advisable to go into extensive details concerning the cystoscopic picture in these cases. There is, however, one misconception that should be corrected. It is generally thought that the urine laden with bacilli infects the mucosa of the bladder and produces ulcerations which lead to exudation. This is highly doubtful. It is more likely a lymphatic submucous involvement with production of tubercles, and these gradually break down into ulcers. I have excised such tubercles from the lips of a ureter meatus and weeks later prior to nephrectomy found the excised areas smoothly covered with healthy mucous membrane, which would hardly have happened if the urine infected the mucosal surface as usually thought.

In the typical cases the specialist will be able to clarify the clinical picture and confirm the diagnosis. Rarely he will have to resort to other aids than those already mentioned, such as the roentgen ray of the urinary tract which in advanced cases may show very definitely, irregularly thickened or calcified areas in the kidney region. Pyelography, I believe, is contraindicated and unnecessary in these cases. It is still a moot question whether diagnostic doses of old tuberculin should be used in particularly obscure cases. There is no doubt that indiscriminate use of tuberculin may harm. On my service it is used only as the last resort prior to operation and occasionally has given valuable information both by increasing the output of bacilli, which had not been found prior to the injection of tuberculin, and by producing a focal reaction in the diseased organ accompanied by fever. With the general improvement in cystoscopic and laboratory work the use of tuberculin has become less and less frequent, and in the future it will be used exceptionally.[7]

Despite the most careful preoperative study one occasionally is in doubt as to whether one is dealing with a renal tuberculosis, and as the operative indication in this disease is a nephrectomy, one must confirm the diagnosis on the operating table. Today many of these cases come to the operating table at a stage when the microscopic external appearance of the kidney is that of a normal organ. If the ureter is thickened it is highly suggestive in a case of sterile renal pyuria with negative roentgen ray, that one is dealing with a tuberculous kidney, the history having been typical. Still before removing such an organ it may be necessary to do a nephrotomy and prove the presence of a tuberculous lesion (and exclude a uric acid calculus). Again, in some particularly difficult cases it may be necessary to expose both kidneys and by palpation of the ureter and examination of both kidneys decide which is diseased or whether both are diseased. With experience in cystoscopic work it is becoming less and less necessary to make the diagnosis on the operating table under either one of the above conditions. In recent years careful preoperative study has invariably sufficed to clarify the diagnosis.

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[7] Beer, E.: Use of Tuberculin in the Diagnosis of Obscure Conditions in the Genito-urinary System, New York Med. Record, October 11, 1913.

Treatment. As renal tuberculosis may be part of a more general tuberculosis, before advising the only treatment that is of avail, namely, nephrectomy, it is essential to make a most thorough examination of the patient. Patients with pulmonary tuberculosis should be operated upon under gas-oxygen. If the disease in the chest is advanced and active operation is inadvisable. The same anesthesia should be used in decrepit or weakened patients. Spinal anesthesia will probably meet the same indications. I have used it successfully in this type of case.

In about 10 per cent of our cases the disease of the ureter has been very extensive producing strictures, in these the aseptic nephroureterectomy described recently[8] was rather regularly done. It is very striking to see how all the irritative symptoms cease directly after the operation and how the urine becomes clear at once. In the other 90 per cent we do a typical nephrectomy—remove a few inches of ureter, carbolize the stump, drop it back and close the wound with a tube drain. No great advantage has been seen from filling the wound with saline and closing it completely. In a series of 100 cases in which nephrectomy was done the mortality has been 2 per cent, and but for an error in interpretation it might well have been half of that. Evidently the operative risk is not serious.

Many series of end-results have been published. Braasch's[9] monumental work is the most recent publication of a large series from the Rochester Clinic, and it deserves careful study. His data show complete cure in 60 per cent; recovery 80 per cent; *i. e.*, deaths within five years of operation, 20 per cent. This corresponds fairly well with the experience of others. Patients who can take care of themselves have a better chance than the laboring classes. About 70 per cent of the former seem to be definitely cured and perhaps 50 to 55 per cent of the less fortunately placed. It is more than likely that with earlier recognition of this disease earlier operation will give even better end-results. If we turn to the end-results of non-operated cases as presented to the German Urological Society by Wildbolz (p. 108) we find that in 316 non-operated cases only 20 per cent lived longer than five years.[10]

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[8] Beer, E., and Hyman, A.: Progress in Nephrectomy, Jour. Am. Med. Assn., October 30, 1920, vol. lxxv.

[9] Surgical Renal Tuberculosis: The Prognosis, Am. Jour. Med. Sc., January, 1920. Verhandl. d. Deutschen Gesellschaft f. Urologie, 1912.

Such data teach very definitely that a tuberculous kidney should be removed as soon as the diagnosis is made and while the disease is still unilateral. Today in close to 10 per cent of the cases when first examined the disease involves both kidneys, pus and tubercle bacilli being definitely found in the secretion of each organ. If nephrectomy of the more diseased organ is done I have never seen any real gain to the patient, and it is my belief that the cases in which marked improvement has been reported may not have been bilateral renal tuberculosis. In a paper on the significance of ureteral tubercle bacilluria.[11] I have called attention to some of the factors which may underly a mistaken diagnosis of bilateral tuberculous disease when the disease is unilateral. Moreover, when both organs are definitely diseased it may be difficult to decide which one is causing more trouble and which one is more diseased.

Functional tests will not regularly help in making this distinction. If one organ delivers practically pure pus and little urea, and is, moreover, a useless sac producing disturbance, its removal may benefit the patient even though the second kidney be diseased and its tuberculosis be uninfluenced. Braasch reports 13 deaths within one and a half years following nephrectomy in 16 patients with bilateral disease, and in a series of 62 cases Israel found that about 70 per cent had died shortly after the nephrectomy.

After the removal of the kidney in unilateral involvement the patient unfortunately is not cured and well. There is always some tuberculosis left in the urinary tract which Nature usually takes care of, provided the patient leads a well-regulated existence. The general hygiene of these patients must be supervised and their resistance carefully built up by nutritous diet and fresh air.

Another difficulty which these patients present is the formation of a sinus (in at least 10 per cent of cases) in the lumbar wound which may take months to heal. These sinuses seem to develop in a definite number of cases even though the operation has been absolutely aseptic and no tuberculosis material has been split. These sinuses are usually spoken of as ureter sinuses, but I believe this is a misnomer, as I have seen them repeatedly even when the ureter and kidney have been removed in one piece. At times the whole lumbar wound breaks down with tuberculosis some five or six weeks after the nephrectomy, and then has to be laid wide open. My experience has suggested to me that we are dealing in these slow-healing postoperative wounds with a tuberculosis due to a tubercle bacteremia induced by the operative manipulations. Attempts to pick up the tubercle bacillus in the circulation during the operation have, however, regularly failed despite the use of guinea-pigs. Improved or modified technic will probably be more successful.

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[10] Wildbolz: Ibid.

[11] Vide ante.

Another condition that may seriously trouble these patients after nephrectomy is the persistence of bladder symptoms with or without hematuria. Fortunately these symptoms gradually disappear in the majority of cases, but in others they plague the patient and they can see no benefit following the operation. All sorts of remedies have been suggested for these cases from Rovsing's carbolic acid to Marion's lactic acid bacillus milk injections. Personally I have seen the best results when using sandalwood oil and cystoscopic high-frequency cauterization of the usually extensive bladder ulcers.

Before closing this brief and rather superficial review of the most striking features of chronic renal tuberculosis, I must say a word about the complications that develop in other organs in those patients that die either after the operation or unoperated. Experience has shown that after nephrectomy those patients who are destined to die develop complications usually within a year of the operation and die as a result of these complications. Whether the dormant processes in other viscera are activated by the operative procedure or whether the operative trauma produces a bacillemia has not been proved. Both factors probably play a part. The frequency of a subacute miliary tuberculosis following the operation, in the 20 per cent that do not live longer than two years must be borne in mind. We have seen several cases of tuberculous meningitis within a few months of the nephrectomy, but as autopsies were not performed it was not known whether this was part of a miliary tuberculosis or not. Careful studies have shown that pulmonary tuberculosis, tuberculosis of the second kidney often unrecognized at the time of operation and miliary tuberculosis contribute to the death-toll during the first and second years following the nephrectomy. In those cases of renal tuberculosis which for one reason or another did not come to operation, the data at hand suggest that renal insufficiency, general amyloid disease due to chronic suppuration and pulmonary tuberculosis lead to the fatal issue. Miliary tuberculosis seems to play less of a role than in the postoperative cases. This naturally leads to the conclusion that in doing a nephrectomy one should traumatize as little as possible, so as to avoid driving bacilli into the circulation.

If during the next generation the developments in the surgery of renal tuberculosis progress as remarkably as they have during the past twenty years, patients will regularly come to the operating table in the earliest stages of their trouble and their chances of permanent relief will thereby be markedly improved. As we have seen the operative risk is low, and the mortality during the first years after the operation is still much too high. Earlier diagnosis and earlier operation followed by better postoperative care will surely lead to better end-results, and with generous coöperation we look forward to a future accomplishment of which we all shall be proud.